situated in the so-called peritoneal cavity. Normally there is no such thing as a peritoneal cavity or intra-peritoneal organ.

Taking, for instance, a loop of small intestine. It is enveloped by peritoneum, but not completely so, since it is attached to the posterior abdominal wall by a mesentery composed necessarily of two layers of peritoneum between which are situated blood vessels, nerves, glands, fat in various quantities and connective tissue. These structures occupy space and therefore prevent the absolute approximation of the two layers of peritoneum, and hence cannot completely envelop the gut. Is it not a fact that if the peritoneal membrane were not so thin, and not so intimately adherent to the organs and parieties, it would be quite possible for one to shell out, as it were, every abdominal organ and inflate again the peritoneal sack to its original shape? If this were possible, it is evident that all abdominal organs would again be found external to the peritoneal sack, proving conclusively that no organ is situated within the sack, and hence cannot be even anticipated intra-peritoneal.

Similar misinterpretations do we find in the so-called plural cavity or sack, in the thorax and conjunctival sack in the orbit of the eye. In both instances the visceral and parietal layers of the respective membranes are so intimately related and so closely approximated that it is impossible, more or less comfortably, or without any difficulty, to introduce into these so-called cavities an object even as thin as tissue paper. Here, too, only when a pathological lesion takes place that a cavity is created in each instance, or on a normal cadaver by a bad dissector. By a cavity we understand a space normal or abnormal not occupied by anything except perhaps air, and that can be filled by fluids or solids as desired. Is there anything in the above mentioned instances corresponding to such a cavity?

In topographical or relational anatomy I find similar misinterpretations. By the relations of an organ or a structure we should include only such organs or structures as are situated in immediate vicinity to the one in question, and if several are so situated, say in front of the one under consideration, it is only that one which is almost in direct contact that we can properly consider in relation to it. As an instance, I will offer the relations of the second and third portions of the subclavian artery. These are situated on the superior surface of the first rib. That the first rib is below and rather at a posterior aspect to the clavicle no one can dispute. Furthermore, to the anterior border of the clavicle the pectoralis major and deltoid muscles are attached and the subclavius beneath the clavicle.

Now the platyzma takes its origin by thin, fibrous bands from the fascia covering the upper part of both pectoralis major and deltoid muscles and its fibers pass over the clavicle obliquely upwards and inwards to be inserted into the subcutaneous tissue of the lower part of the face, blending with the muscles of that region. Since the platyzma is situated in front of the pectoralis major and the subclavian artery on the first rib, it is clear that the vessel is covered not only by this muscle but also by the pectoral fascia, costocoracoid membrane and subclavian muscle. The platyzma is consequently most anterior in the upper pectoral region. How then can the platyzma be considered as a structure in relation with the subclavian artery?

Again, if we interpret this to our students as one of the relations of the subclavian artery, what would be the objection of teaching them also that the umbilicus is in anterior relation to the abdominal aorta? I have examined as many text-books on anatomy as I could possibly obtain, and, with few exceptions, the platyzma is mentioned as one of the relations of the vessel under discussion. I have made careful, repeated dissections of that region, and I have also very carefully investigated the dissections made by my students, and yet fail to see the veracity of that statement. I can only conclude that it is a misinterpretation, unreasonable and confusing to the student.

In my didactic course I invariably point out to my students this as well as all other similar misinterpretations, as I call them. Time and space will not permit me to present in detail all investigations I have made in this respect, but I hope in future to be able to present further results in the field of gross anatomy and I simply offer this paper as an impetus for discussion.

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## TRANSFUSION IN A CASE OF TYPHOID FEVER.

By RENÉ BINE, M. D., San Francisco.

In his book "Hemorrhage and Transfusion" Crile reports two instances of intestinal hemorrhages complicating typhoid fever where transfusion produced marked improvement. Both patients were remarkably revived, but the hemorrhages recurred and death resulted. Crile has likewise collected ten other cases of typhoid fever with intestinal hemorrhages where transfusion of blood (whole blood 2, defibrinated 4, not stated but probably defibrinated 4) apparently saved five lives. These cases date back to a period of from 1875 to 1886, when direct transfusion was unknown, so that in view of the great rarity of these reports, we feel justified in publishing an account of the following case which presented many interesting features, and in which, we feel, transfusion accomplished all that could be expected of it under the circumstances.

Miss F. C., born 1882, single; nurse.

Family history: M. d. acute t. b. 1885 f. and 4 s. alive and well.

Habits: Irregular hours food. Otherwise negative. Menses regular.

Past history: Pneumonia at 12 years age. Diphtheria, pertussis in childhood. Tonsillitis several times, tonsils removed 1905. Following a period of very hard work in 1908 was quite anemic (Reds 3,100,000 Hg. 70%) and exceedingly nervous, had frequent crying spells, and at this time examination revealed a cardiac murmur (haemic?) and a slight albuminuria, the latter clearing up and the patient gaining in weight under tonic treatment. The patient never returned for examination and except for more or less constipation and spells of nervous exhaustion due to overwork, was free from trouble until this:

Present Illness: Having been on night duty for several weeks and unable to sleep during the day on account of nervousness the patient on December

22, 1910, complained of being "all in," feeling nervous and "weepy" and of a very severe headache. Temperature 99 degrees. By the 24th the temperature had risen to 102 degrees, the area of splenic dullness was distinctly enlarged, rose spots were present, and by the 27th a diazo reaction and a weak Widal reaction were obtained. The patient was in a rather undernourished state, quite nervous and impressed the observer as profoundly toxic.

Course of the Disease: The patient's chief complaint was headache, the pain being so severe as to interfere with her rest and was not relieved by ordinary doses of acetphenetidin. Ice-bags to the head, cold sponges and hexamethylenamine internally were employed to combat the pyrexia and undoubted bacillaemia.

December 25: Temperature ranged from 103 to 104.6, pulse from 104 to 120, respirations 18 to 22. Leucocytes 7800, polys 67%. General chilliness was frequently complained of.

December 26: Temperature 102 to 104.8. Continual nausea, repeated vomiting, frequent chilliness; rectal pains and tenderness partly due to hemorrhoids.

December 27: Temperature 100.8 to 105, pulse 100 to 118. Frequent nausea, chilliness, "feels miserable," headache persists, urine examination negative except for marked diazo.

December 28: Moderately comfortable for a few hours, then chilliness, vomiting twice, considerable nausea, loose bowels and pain in right upper quadrant abdomen. This region is tender, there is marked rigidity, and a definite resistance to palpation. Temperature 101 to 104.6.

December 29: Temperature 101.6 to 104.8. Very restless and nervous. Headache persists. Late in day, nose bleed. Urine as on 27th. Still has abdominal pain.

December 30: Temperature 101.2 to 104. Nausea, vomiting, constipation and distention relieved by 15 cc. castor oil.

December 31: Severe headache and moderate backache persist. Sponges fatigue patient greatly. Nausea present. Abdomen still painful and tender. Gall-bladder can be distinctly felt, descending apparently about 1" below liver, which is quite enlarged. In order to reduce temperature and combat headache, pyramidon prescribed, 10 cc. of a 2% solution every 2 hours. Temperature 101 to 103.4.

January 1st: Gas pains have bothered patient from onset of trouble. Has been subject to them before this illness, as well as to pains in rectum, which are only relieved by low cold water enemas. Vomiting and nausea. 4 p. m. about 120 cc. liquid stool highly colored with blood. 8 p. m., coffee-ground vomitus giving positive reaction to all blood tests. Temperature fell to 100° after bowel hemorrhage but did not rise above 101.2 during remainder of day.

January 2nd: Temp. 100.4 to 103.4. Pulse 100 to 130. Leucocytes 7200. Polys 85%. Cholecystitis still persists. Severe pain in rectum; low cold water enemas insisted on by patient, return highly colored with blood. Urine contains much albumin, many hyaline, granular and epithelial casts, but no blood.

January 3: Temp. 100 to 101.2. Patient getting weaker and is unable to retain but a very small amount of ingested liquids. Headache less since taking pyramidon. Vomited blood; low enema returned bloody.

January 4: Still blood per rectum. Turpentine stupes. Patient quite weak. Vomiting of blood, bright red, also bright red blood in stools and epistaxis. Gums have shown decided tendency to bleed from onset of illness. Temp. 100.2 to 102.2.

January 5: Rectal examination (digital and with proctoscope just inside sphincter) fails to show any source of bleeding. This examination was undertaken to determine if hemorrhoids were responsible for blood in stools. Gelatine fed patient. Pyramidon discontinued in view of a possible influence on hemorrhagic tendency, and hexamethylenamine gr. v. every four hours prescribed. Temp. 101 to 102.6. Pulse 100 to 110. Resp. 20 to 24. Widal quite marked.

January 6: Menses present. 11 a. m. Large bowel movement of almost pure blood; pulse weak, 124. 11:20 a. m. Small bloody stool. Two stools together equal about 500 cc. Morphine hypodermically; coil to abdomen. 3 p. m. severe chill. 6:45 p. m. coffee-ground vomitus. Leucocytes 7000, polys. 60%. Hemoglobin 70%. Calcium chloride enemas. Temp. 103.2 to 103.8. Pulse 112 to 124.

January 7: Temp. 102.4 to 103.6. Less nausea, able retain moderate amount liquid nourishment. Chilly and quite nervous during day. Sleep about 6 hours in 24.

January 8: Ice-coil to abdomen has been repeatedly employed but poorly tolerated, generally producing marked chilliness. Several chills to-day and repeated vomiting. Temp. 103.4 to 104.6. Pulse 104 to 120.

January 9: Temp. 103.8 to 104.4. Complains of nervousness. Two chills during day, one quite strong.

January 10: Chilliness. 8 a. m. Bowel movement; apparently large proportion bright red blood mixed with many small clots and small amount urine; total measured 600 cc. Morphine hypo. 8:20 a. m. 15 cc. pure blood per rectum. Temp. 103.4. Pulse 118. Resp. 24. Calcium Chloride enema. Gelatins. Complains of queer sensation about heart; pulse rapid but fair quality. 12 m. temp. 101.8. Pulse 124. Resp 22. 12:20 p. m. vomited 12:30 p. m. large bowel hemorrhage. Morphin hypo. Adrenalin m x hypo. Adrenalin enema. 3:30 p. m. medium large hemorrhage. Pulse 150. Morphin and adrenalin hypo. 4:20 p. m. Large bowel homorrhage. 5:20 p. m. Temp. 100.9. Pulse 146. Quite weak. 6 p. m. pulse 160. Patient's condition appears desperate. For days she has retained practically nothing but liquids. Is now exceedingly weak, pulse barely palpable, rapid, thready, and the usually pale face appears ghastly white and old, the features drawn. Red blood cells 2,072,000. Hemoglobin 35%, leucocytes 7100.

The patient's condition certainly seemed hopeless. The homorrhages were far greater than one could expect with an ordinary typhoid. Gastric hemorrhages as well as the early epistaxis and bleeding gums pointed to a general septic condition, though ulcers in the stomach could not be definitely ruled out.

It was decided to do a transfusion immediately. It was naturally impossible to carry out hemolytic tests with prospective donors, nor could time be lost in searching for a person who had previously had typhoid, to act as donor. A nurse was found who volunteered to make the sacrifice on ten minutes notice, and at 7:10 p. m. the patient was taken to the operating room, where Wallace I. Terry and Sterling Bunnell carried out the direct method of transfusion.

The result was soon noticeable; the patient's pulse became fuller, the great anxiety and dyspnoea slightly lessened and the pearly conjunctiva assumed a better color and the wrinkles about the mouth became less apparent. But while the transfusion was still progressing, matters again seemed to take a turn for the worse, so that at this time it was assumed that another hemorrhage was taking place.

The blood count before the transfusion was 2,007,200 with 35% hemoglobin; 2,340,000 with 40% hemoglobin after the transfusion. The patient suffered a severe chill as soon as she was returned to her bed from the operating room. Pulse 140. Resp. 28. Temp. 104.8 (rectal).

January 11: At 1:15 a. m. large bowel hemorrhage. 1:30 a. m. vomited. Vomiting of all nourishment and liquids during entire day. Headache. Temp. range 97.8 to 99 (rectal). Pulse 104 to 114. Resp. 18 to 22. Urine contained large amount of albumin but no blood. Diazo still present.

January 12: Difficulty in breathing, only in part due to abdominal distention, but while of sighing type, no increase in rate, not relieved by oxygen inhalation. Vomited 14 times in 24 hours. Temp. 97 to 100 (rectal). Pulse 102-14. Resp. 18 to 20. Urine 250 cc.

January 13: Ringer's solution subcutaneously, and salt solution per rectum. Vomited 3 times. Only 100 cc. urine, this per catheter. Patient decidedly weaker. Temp. 98.4 to 99.4 (rectal). Pulse 100 to 108. Resp. 15-20.

January 14: Nutrient enemas. Nausea and vomiting persist. Temp. 98.2 to 98.4 (rectal). Pulse 94-104. Urine 150 cc. (catheter). One enema was returned with some old clotted blood.

January 15: Temp. 98.2 to 98.6 (rectal). Pulse 96 to 104. Urine 120 cc. (catheter). Vomiting persistent.

January 16: Very weak. Temp. 97.2 to 97.4 (rectal). Urine 125 cc. (catheter). At times seems to be in stupor.

January 17: Temp. 96.6 to 97 (rectal). Pulse 92 to 96. Resp. 16 to 22. Urine 270 cc. (catheter). Parotid glands are tender and a trifle enlarged.

January 18: Quite weak and exhausted. Temp. 96.6 to 98 (rectal). Urine 150 cc. (catheter). During last 2 days, whatever patient desired was given her, steak to chew, custard, etc., but within a half hour it was always vomited. Has spells of difficult breathing. Parotid glands are very tender and palpably swollen.

January 19: Temp. 96. Pulse 120. Resp. 30-40. Constant difficult breathing unrelieved by oxygen inhalations. Pain around heart complained of. Leucocytes 24,000.

During the last few days a mass had been felt in the right flank further down than the gall-bladder. which has decreased gradually in size. It seemed too large for kidney, and in view of a possible perirenal abscess (pyo-nephritis practically excluded by absence of albuminuria, this having disappeared a few days ago, and by absence of renal elements in sediment, the pus cells being easily explainable by a moderate degree of cystitis; sediment showed presence of bacilli, culturally typhoid), needles were inserted in flank but without finding pus, this under nitrous oxide anesthesia with patient in her own bed. After this, slept at short intervals, waking up to complain of inability to get breath. Morphin relieved this distress, and becoming gradually weaker, conscious practically to the very end, patient died on January 20 at about 7:30 a. m.

The body having been embalmed very soon after death, it was possible to perform but an incomplete and somewhat unsatisfactory autopsy. The abdomen only was opened. The embalmer's needle had punctured the gall-bladder to such an extent as to warrant no deductions as to size. The gastric mucosa was injected in a few spots, was for the most part hyperaemic, but no ulcerations were seen. The intestine showed typical typhoid ulcers in the ileo-caecal region, most of them in process of healing.

Large masses of blood clot occupied the lumen of the bowel in this neighborhood, much of which must have been from recent hemorrhage. The kidneys were of the large white variety, (parenchymatous nephritis), the right one reaching down to the anterior superior spine. It measured 14 by 6½ by 4½ cms., the left one 12 by 6 by 5 cms.

We can but feel that the transfusion accomplished something, in fact far more than the blood counts indicated, for the patient no doubt had a hemorrhage while on the operating table, and this helped to lower the post-operative count. We believe that without the transfusion death would have occurred on January 10th instead of ten days later. The remarkable feature of the case was the fact that, following the transfusion, the patient's temperature never went beyond the normal, although on the ninth it had been up to 104.4 (rectal). This cannot be attributed to shock nor to the anemia, for almost up to the last, the pulse was of such character as to exclude these possibilities.

We are of the opinion that the patient's chronic parenchymatous nephritis had much to do with her hemorrhagic tendency; that the latter was aggravated by her severe typhoid; that both combined, but principally the former were responsible for the persistent vomiting. It is curious enough that the albuminuria diminished a few days after the transfusion.

Should we ever again be called upon to treat a severe case of typhoid complicated by a hemorrhagic tendency, we would if possible procure a donor immune to typhoid, and whose serum, tested for hemolysins and iso-agglutinins, was proved devoid of danger to the patient.

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## HOSPITAL DEPARTMENT — SAN FRANCISCO HOSPITALS.

By WM. R. DORR, M. D., San Francisco.

Perhaps no other city in history and certainly no other modern city has had more incentive to construct and put in running order a large number of hospitals than San Francisco has had during the last five years, and I am sure that no community could have responded more loyally to the need than has been done during this more or less trying period.

In the spring of 1906 San Francisco had about forty hospitals, of which very few could be described as thoroughly modern and up to date.

**Private Hospitals.**

| | No. | Beds. |
|---|---|---|
| Hospitals for special classes of cases. using mostly remodeled buildings..... | 7 | 152 |
| General hospitals using old buildings or buildings not originally intended for hospital purposes ..... | 8 | 500 |
| General hospitals using modern buildings built for hospital purposes but over five years old..... | 4 | 600 |
| General hospitals having buildings constructed for hospital purposes during the last five years..... | 11 | 1180 |
| | 30 | 2432 |

**Municipal Hospitals.**

| | No. | Beds. |
|---|---|---|
| Emergency Hospitals..... | 5 | 49 |
| Detention Hospital..... | 1 | 8 |
| Smallpox and Leper Hospitals..... | 2 | 70 |
| General Hospital..... | 1 | 350 |
| General Hospital (in process of construction) ..... | 1 | 510 |
| Tubercular Hospital (planned)..... | 1 | 260 |
| Infectious Hospital (planned)..... | 1 | 100 |
| | 12 | 1347 |

This gives a grand total of 3779 beds or one bed to about every 110 inhabitants.

After the great conflagration only four hospitals